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* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  AUG 09   INSPEC enhanced with 1898-1968 archive
 NEWS  4  AUG 28   ADISCTI Reloaded and Enhanced
 NEWS  5  AUG 30   CA(SM)/CAplus(SM) Austrian patent law changes
 NEWS  6  SEP 11   CA/CAplus enhanced with more pre-1907 records
 NEWS  7  SEP 21   CA/CAplus fields enhanced with simultaneous left and right
                   truncation
 NEWS  8  SEP 25   CA(SM)/CAplus(SM) display of CA Lexicon enhanced
 NEWS  9  SEP 25   CAS REGISTRY(SM) no longer includes Concord 3D coordinates
 NEWS 10  SEP 25   CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
 NEWS 11  SEP 28   CEABA-VTB classification code fields reloaded with new
                   classification scheme
 NEWS 12  OCT 19   LOGOFF HOLD duration extended to 120 minutes
 NEWS 13  OCT 19   E-mail format enhanced
 NEWS 14  OCT 23   Option to turn off MARPAT highlighting enhancements available
 NEWS 15  OCT 23   CAS Registry Number crossover limit increased to 300,000 in
                   multiple databases
 NEWS 16  OCT 23   The Derwent World Patents Index suite of databases on STN
                   has been enhanced and reloaded
 NEWS 17  OCT 30   CHEMLIST enhanced with new search and display field
 NEWS 18  NOV 03   JAPIO enhanced with IPC 8 features and functionality

 NEWS EXPRESS  JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN    Welcome Banner and News Items
 NEWS IPC8     For general information regarding STN implementation of IPC 8
 NEWS X25      X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that
specific topic.

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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 10:41:55 ON 06 NOV 2006

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
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FULL ESTIMATED COST                                    0.21        0.21

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
      AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
      CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
      DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 10:42:11 ON 06 NOV 2006

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s (adenylate(w)cyclase) and (nonsense)
           3   FILE AGRICOLA
           1   FILE BIOENG
           5   FILE BIOSIS
           7   FILE BIOTECHNO
          19   FILE CAPLUS
           1   FILE DRUGU
           1   FILE EMBAL
           8   FILE EMBASE
           1   FILE ESBIOBASE
          19   FILE GENBANK
  35 FILES SEARCHED...
           4   FILE LIFESCI
           8   FILE MEDLINE
           4   FILE PASCAL
          10   FILE SCISEARCH
           2   FILE TOXCENTER
         197   FILE USPATFULL
          18   FILE USPAT2
  66 FILES SEARCHED...

  17 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L1   QUE (ADENYLATE(W) CYCLASE) AND (NONSENSE)

=> file scisearch medline caplus
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                               1.22       1.43

FILE 'SCISEARCH' ENTERED AT 10:43:18 ON 06 NOV 2006
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FILE 'MEDLINE' ENTERED AT 10:43:18 ON 06 NOV 2006

FILE 'CAPLUS' ENTERED AT 10:43:18 ON 06 NOV 2006
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=> s (adenylate(w)cyclase) and (nonsense)
L2          37 (ADENYLATE(W) CYCLASE) AND (NONSENSE)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           26 DUP REM L2 (11 DUPLICATES REMOVED)

=> d l3 1-26 ti

L3   ANSWER 1 OF 26  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Genes showing altered levels of expression in pancreatic disease and their
     use in diagnosis and prognosis of pancreatic cancer
```

L3 ANSWER 2 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 1
TI Development of a Premature Stop Codon-detection method based on a bacterial two-hybrid system

L3 ANSWER 3 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Development of a premature stop codon-detection method based on a bacterial two hybrid system

L3 ANSWER 4 OF 26 MEDLINE on STN
TI Characterization of four receptor cDNAs: PAC1, VPAC1, a novel PAC1 and a partial GHRH in zebrafish.

L3 ANSWER 5 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
TI Gene expression profiles and biomarkers for the detection of lung disease-related and other disease-related gene transcripts in blood

L3 ANSWER 6 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Knock-down of RGS(4) and beta tubulin in CHO cells expressing the human MT1 melatonin receptor prevents melatonin-induced receptor desensitization

L3 ANSWER 7 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Insight into the genome of Aspergillus fumigatus: analysis of a 922 kb region encompassing the nitrate assimilation gene cluster

L3 ANSWER 8 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Pharmacological characterization of β-endorphin- and dynorphin A1-17-induced feeding using G-protein α-subunit antisense probes in rats

L3 ANSWER 9 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI GNAS1 lesions in pseudohypoparathyroidism Ia and Ic: genotype phenotype relationship and evidence of the maternal transmission of the hormonal resistance

L3 ANSWER 10 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Feeding induced by food deprivation is differentially reduced by G-protein α-subunit antisense probes in rats

L3 ANSWER 11 OF 26 MEDLINE on STN
TI Expression of subunits for the cAMP-sensitive 'olfactory' cyclic nucleotide-gated ion channel in the cochlea: implications for signal transduction.

L3 ANSWER 12 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Transcriptional profiling reveals global defects in energy metabolism, lipoprotein, and bile acid synthesis and transport with reversal by leptin treatment in Ob/ob mouse liver

L3 ANSWER 13 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Absence of constitutively activating mutations in the GHRH receptor in GH-producing pituitary tumors

L3 ANSWER 14 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 3
TI Deficiency of the alpha-subunit of the stimulatory G protein and severe extraskeletal ossification

L3 ANSWER 15 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 4
TI Characterization of a new set of mutants deficient in fermentation-induced loss of stress resistance for use in frozen dough applications

L3 ANSWER 16 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on

STN
TI Morphine and morphine-6 beta-glucuronide-induced feeding are differentially reduced by G-protein alpha-subunit antisense probes in rats

L3 ANSWER 17 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Gene probes used for genetic profiling in healthcare screening and planning

L3 ANSWER 18 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Gene probes used for genetic profiling in healthcare screening and planning

L3 ANSWER 19 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 5
TI The effects of antisense to G(i alpha 2) on opioid agonist potency and G(i alpha 2) protein and mRNA abundance in the mouse

L3 ANSWER 20 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Functional rescue of mutant V2 vasopressin receptors causing nephrogenic diabetes insipidus by a co-expressed receptor polypeptide

L3 ANSWER 21 OF 26 MEDLINE on STN
TI The effect of eight V2 vasopressin receptor mutations on stimulation of adenylyl cyclase and binding to vasopressin.

L3 ANSWER 22 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI MOLECULAR-BASIS OF FAMILIAL GROWTH-HORMONE DEFICIENCY

L3 ANSWER 23 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI CHARACTERIZATION OF THE CYR1-2 UGA MUTATION IN SACCHAROMYCES-CEREVISIAE

L3 ANSWER 24 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 6
TI GENETIC AND MOLECULAR ANALYSES OF THE SUP201 GENE - A TRANSFER RNA3ARG NONSENSE SUPPRESSOR OF YEAST CYR1-2

L3 ANSWER 25 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 7
TI IDENTIFICATION OF THE STRUCTURAL GENE AND NONSENSE ALLELES FOR ADENYLATE-CYCLASE IN SACCHAROMYCES-CEREVISIAE

L3 ANSWER 26 OF 26 MEDLINE on STN DUPLICATE 8
TI Cyclic AMP may not be involved in catabolite repression in Saccharomyces cerevisiae: evidence from mutants unable to synthesize it.

=> d l3 1 2 13 21 ti abs bib

L3 ANSWER 1 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer
AB Genes showing altered levels of expression in healthy vs. neoplastic pancreas are identified for use in the diagnosis of cancers including ductal adenocarcinoma; as indicators in screening for effective drugs; and as targets for nucleic acid-based therapies including antisense nucleic acids or siRNA. Gene expression profiling identified 1419 genes showing changes in levels of expression in neoplastic epithelium of which 650 were up-regulated and 769 were down-regulated. Of the 1419 genes, 1267 were not previously known to have any connection with pancreatic neoplasms.
AN 2006:238155 CAPLUS
DN 144:310062
TI Genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer

IN Kloeppel, Guenter; Luettges, Jutta; Kalthoff, Holger; Ammerpohl, Ole; Gruetzmann, Robert; Pilarsky, Christian; Saeger, Hans Detlev; Alldinger, Ingo
PA Technische Universitaet Dresden, Germany
SO Ger. Offen., 132 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 102004042822	A1	20060316	DE 2004-102004042822	20040831
WO 2006024283	A2	20060309	WO 2005-DE1527	20050826
WO 2006024283	A3	20060831		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI DE 2004-102004042822 A 20040831
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 26 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 1
TI Development of a Premature Stop Codon-detection method based on a bacterial two-hybrid system
AB Background: The detection of Premature Stop Codons (PSCs) in human genes is very useful for the genetic diagnosis of different hereditary cancers, e. g. Familial Breast Cancer and Hereditary Non-Polyposis Colorectal Cancer (HNPCC). The products of these PSCs are truncated proteins, detectable in vitro by the Protein Truncation Test and in vivo by using the living translation machinery of yeast or bacteria. These living strategies are based on the construction of recombinant plasmids where the human sequence of interest is inserted upstream of a reporter gene. Although simple, these assays have their limitations. The yeast system requires extensive work to enhance its specificity, and the bacterial systems yield many false results due to translation re-initiation events occurring post PSCs. Our aim was to design a recombinant plasmid useful for detecting PSCs in human genes and resistant to bacterial translation re-initiation interferences.

Results: A functional recombinant plasmid (pREAL) was designed based on a bacterial two-hybrid system. In our design, the in vivo translation of fused fragments of the Bordetella pertussis adenylate cyclase triggers the production of cAMP giving rise to a selectable bacterial phenotype. When a gene of interest is inserted between the two fragments, any PSC inhibits the enzymatic activity of the product, and translation re-initiation events post-PSC yield separated inactive fragments. We demonstrated that the system can accurately detect PSCs in human genes by inserting mutated fragments of the brca1 and msh2 gene. Western Blot assays revealed translation re-initiation events in all the tested colonies, implying that a simpler plasmid would not be resistant to this source of false negative results. The application of the system to a HNPCC family with a nonsense mutation in the msh2 gene correctly diagnosed wild type homozygous and heterozygous patients.

Conclusion: The developed pREAL is applicable to the detection of PSCs in human genes related to different diseases and is resistant to

translation re-initiation events. The diagnosis steps are easy, have a low cost, detect only pathologic mutations, and allow the analysis of separated alleles.

AN 2006:932986 SCISEARCH
GA The Genuine Article (R) Number: 086PV
TI Development of a Premature Stop Codon-detection method based on a bacterial two-hybrid system
AU Real S M; Marzese D M; Gomez L C; Mayorga L S; Roque M (Reprint)
CS Natl Univ Cuyo, Cellular & Mol Lab, Fac Med Sci, IHEM, Mendoza, Argentina (Reprint)
sreal@unsl.edu.ar; marzese.diego@fcm.uncu.edu.ar; lgomez@fcm.uncu.edu.ar; lmayorga@fcm.uncu.edu.ar; mroque@fcm.uncu.edu.ar
CYA Argentina
SO BMC BIOTECHNOLOGY, (2 SEP 2006) Vol. 6, arn. 38.
ISSN: 1472-6750.
PB BIOMED CENTRAL LTD, MIDDLESEX HOUSE, 34-42 CLEVELAND ST, LONDON W1T 4LB, ENGLAND.
DT Article; Journal
LA English
REC Reference Count: 31
ED Entered STN: 18 Oct 2006
Last Updated on STN: 18 Oct 2006
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L3 ANSWER 13 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
TI Absence of constitutively activating mutations in the GHRH receptor in GH-producing pituitary tumors
AB The mol. events leading to the development of GH-producing pituitary tumors remain largely unknown. The authors hypothesized that activating mutations of the GHRH receptor might occur in a subset of GH-producing pituitary tumors. Genomic DNA samples from 54 GH-producing pituitary tumor tissues were screened for mutations of the GHRH receptor. Eleven homozygous or heterozygous nucleotide substitutions [169G > A (A57T), 338C > T (P113L), 363G > T (E121D), 409C > T (H137Y), 547G > A (D183N), 673G > A (V225I), 749G > A (W250X), 760G > A (V254M), 785G > A (S262N), 880G > A (G294R), 1268G > A (C423Y)] were found in 12 patients (22.2%). The 169G > A substitution (A57T) appears to be a polymorphism (4 patients, 7.4%). E121D and V2251 were each found in 2 patients. In 1 patient with the V2251 sequence, the substitution was not found in genomic DNA from peripheral leukocytes, suggesting a somatic mutation. A patient with a heterozygous W250X mutation was homozygous for the C423Y substitution. These variant GHRH receptors were studied in transfected TSA-201 cells to evaluate the functional consequences of the amino acid changes. None of the GHRH receptor variants was associated with basal elevation of intracellular cAMP. GHRH induced variable cAMP responses. With the W250X and G294R variants, there was no cAMP stimulation by GHRH, indicating that the mutations are inactivating. Expression of the W250X GHRH receptor on the cell membrane was severely decreased and GHRH binding to the G294R GHRH receptor was impaired. Although GHRH receptor variants are common in GH-producing pituitary adenomas, constitutively activating mutations, as a mechanism for GH-producing pituitary tumors appear to be rare.
AN 2001:593815 CAPLUS
DN 135:301898
TI Absence of constitutively activating mutations in the GHRH receptor in GH-producing pituitary tumors
AU Lee, Eun Jig; Kotlar, Tom J.; Ciric, Ivan; Lee, Mi Kyung; Lim, Sung Kil; Lee, Hyun Chul; Huh, Kap Bum; Mayo, Kelly E.; Jameson, J. Larry
CS Division of Endocrinology, Metabolism, Northwestern University Medical School, Chicago, IL, 60611, USA
SO Journal of Clinical Endocrinology and Metabolism (2001), 86(8), 3989-3995
CODEN: JCEMAZ; ISSN: 0021-972X
PB Endocrine Society
DT Journal
LA English
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 21 OF 26 MEDLINE on STN
TI The effect of eight V2 vasopressin receptor mutations on stimulation of adenylyl cyclase and binding to vasopressin.
AB We previously identified six V2 vasopressin receptor mutations in five unrelated nephrogenic diabetes insipidus (NDI) families. In order to elucidate the effect of these mutations on the function of the V2 vasopressin receptor, we introduced these six and two additional, naturally occurring mutations into the V2 vasopressin receptor gene by in vitro mutagenesis. Five of the mutants (two frameshift, one nonsense, and two missense) failed to stimulate adenylyl cyclase due to their inability to bind vasopressin under the experimental conditions. In contrast, ligand binding and cAMP accumulation were normal for two other mutations, a A61V missense mutation and an in-frame deletion of four amino acids (Arg-247 to Gly-250), suggesting that they are not the cause of NDI in these families. The deletion mutation was found in a family in conjunction with a second mutation, R181C, which yielded a much reduced ligand-binding capacity. The KD of R181C was at least 26 times higher than that of the wild type. Further characterization by an immunofluorescent assay showed that the R181C mutant receptor is expressed and distributed on the cell surface in a manner similar to that of the wild type. This finding indicates that the inability of this mutant to stimulate adenylyl cyclase is caused by the reduced capacity for vasopressin binding and that the R181C mutation is responsible for NDI in this family.
AN 95081152 MEDLINE
DN PubMed ID: 7527400
TI The effect of eight V2 vasopressin receptor mutations on stimulation of adenylyl cyclase and binding to vasopressin.
AU Pan Y; Wilson P; Gitschier J
CS Howard Hughes Medical Institute, University of California, San Francisco 94143.
SO The Journal of biological chemistry, (1994 Dec 16) Vol. 269, No. 50, pp. 31933-7.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199501
ED Entered STN: 24 Jan 1995
Last Updated on STN: 6 Feb 1998
Entered Medline: 12 Jan 1995

=> s (adenylate(w)cyclase) and (gene(w)therapy)
L4 95 (ADENYLATE(W) CYCLASE) AND (GENE(W) THERAPY)

=> s l4 and nonsense
L5 0 L4 AND NONSENSE

=> s (adenylate(w)cyclase(w)inhibi?) and (gene(w)therapy)
L6 13 (ADENYLATE(W) CYCLASE(W) INHIBI?) AND (GENE(W) THERAPY)

=> d l6 1-13 ti

L6 ANSWER 1 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Therapeutic targeting of parc/ccl18 and its signaling in pulmonary fibrosis

L6 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI rAAV vector for expressing biopacemakers in myocardial cells to decrease the conductance of an ion channel responsible for cellular excitability and for treating cardiac pacing dysfunction

L6 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Inhibitory G protein overexpression provides physiologically relevant heart rate control in persistent atrial fibrillation

L6 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Neuronal and optic nerve gene expression patterns in the optic nerve axotomy model of neuronal degeneration

L6 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Selective target cell activation by expression of a G protein-coupled receptor activated superiorly by synthetic ligand (RASSL) for disease treatment and disease modeling

L6 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Therapeutics for heart failure and aging based on interactions of Myo/V1 with transcription factor NFκB subunits

L6 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Cardiac arrhythmia treatment methods using polynucleotides modulating heart electrical property

L6 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Cloning, characterization and therapeutic use of P2Y12 receptor and association of the mutant P2Y12 with bleeding disorder

L6 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Mammalian gonadotropin-releasing hormone (GnRH) receptor expression cassette and therapeutic uses thereof

L6 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Sequence and expression patterns of human and mouse CNRE binding transcription factors factors with therapeutic applications for renin-angiotensin system disorders involving c-Myc and type II collagen and T-cell receptor expression

L6 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Antisense DNA constructs for expression of hybrid mRNAs driven by inducible tissue-specific promoters

L6 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI Functional coupling of overexpressed β1-adrenoceptors in the myocardium of transgenic mice

L6 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2006 ACS on STN
TI β-Adrenergic linked signal transduction mechanisms in failing hearts

=> s (gene(w)therapy) and arthritis
L7 1990 (GENE(W) THERAPY) AND ARTHRITIS

=> s l7 not py>2004
L8 1426 L7 NOT PY>2004

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 979 DUP REM L8 (447 DUPLICATES REMOVED)

=> s l9 and rheumatoid
L10 638 L9 AND RHEUMATOID

=> d l10 1-20 ti

L10 ANSWER 1 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Stimulation of proteoglycan synthesis by glucuronosyltransferase-1 gene delivery: A strategy to promote cartilage repair

L10 ANSWER 2 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Signal transduction pathways: new targets for treating rheumatoid arthritis

L10 ANSWER 3 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI The 2003 Nicolas Andry Award: Orthopaedic gene therapy

L10 ANSWER 4 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Thrombospondin 2 functions as an endogenous regulator of angiogenesis and inflammation in rheumatoid arthritis

L10 ANSWER 5 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Inflammation-responsive promoters for fine-tuned gene therapy in rheumatoid arthritis

L10 ANSWER 6 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy for autoimmune diseases

L10 ANSWER 7 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Tissue engineering through autologous mesenchymal stem cells

L10 ANSWER 8 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Monoclonal antibody-based genetic immunotherapy

L10 ANSWER 9 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Induction of apoptosis of human osteoclasts by the transcription factor decoy approach: relevance for the treatment of rheumatoid arthritis

L10 ANSWER 10 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Complexity in the vascular permeability factor/vascular endothelial growth factor (VPF/VEGF)-receptors signaling

L10 ANSWER 11 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy for autoimmune diseases: Quo Vadis?

L10 ANSWER 12 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Nonviral gene therapy by electrotransfer of HTNF-alpha soluble receptor-i variants and its application to the treatment of experimental arthritis

L10 ANSWER 13 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy in animal models of rheumatoid arthritis: are we ready for the patients?

L10 ANSWER 14 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI IFN-beta in rheumatoid arthritis

L10 ANSWER 15 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation

on STN
TI Recent developments in molecular therapeutic approaches for rheumatoid arthritis

L10 ANSWER 16 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Protection against collagen-induced arthritis by electrotransfer of an expression plasmid for the interleukin-4

L10 ANSWER 17 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Immunotherapeutic approaches in multiple sclerosis

L10 ANSWER 18 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Methods for targeting biologicals to specific disease sites

L10 ANSWER 19 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Targeted gene therapy: frontiers in the development of 'smart drugs'

L10 ANSWER 20 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Feline immunodeficiency virus vectors for efficient transduction of primary human synoviocytes: Application to an original model of rheumatoid arthritis

=> d l10 2 4 5 6 11 12 13 15 19 ti abs bib

L10 ANSWER 2 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Signal transduction pathways: new targets for treating rheumatoid arthritis
AB Biotherapies and other new treatments introduced over the last few years have considerably enriched the therapeutic armamentarium for rheumatoid arthritis. Nevertheless, primary refractoriness or secondary escape phenomenon may occur, indicating a need for identifying new treatment targets. Promising candidates can be found among compounds involved in signal transduction pathways, most notably protein kinases (mitogen-activated protein kinase, MAPK and phosphatidylinositol-3 protein kinase, PI3) and transcription factors (nuclear factor kappa B. NF-kappaB activating protein 1, AP-1; CCAAT/enhancer-binding protein, C/EB.P and signal transducer and activator of transcription, STAT). Inhibition of signal transduction pathways may be achievable via three main strategies: pharmacological inhibitors, anti-sense or more specific inhibitors such as oligionucleotides or interfering mRNA, and induced overexpression of naturally occurring inhibitors. Clinical trials are under way to evaluate pharmacological inhibitors such as p38 MAPK. Although the preliminary results are promising, proof of safety has not yet been obtained. Signal transduction pathways are involved in normal processes, whose inhibition might produce untoward effects. (C) 2004 Elsevier SAS.
AN 2005:90239 SCISEARCH
GA The Genuine Article (R) Number: 886JS
TI Signal transduction pathways: new targets for treating rheumatoid arthritis
AU Morel J (Reprint); Berenbaum F
CS CHU Lapeyronie Hosp, Immunorheumatol Dept, 371, Ave Doyen Gaston Giraud, F-34295 Montpellier 5, France (Reprint); CHU Lapeyronie Hosp, Immunorheumatol Dept, F-34295 Montpellier 5, France; CHU Lapeyronie Hosp, INSERM, U 454, F-34295 Montpellier, France; Univ Paris 06, St Antoine Hosp, CNRS, Dept Rheumatol, Paris, France; Univ Paris 06, St Antoine Hosp, CNRS, UMR 7079, Paris, France

j-morel@chu-montpellier.fr
CYA France
SO JOINT BONE SPINE, (NOV 2004) Vol. 71, No. 6, pp. 503-510.
ISSN: 1297-319X.
PB EDITIONS SCIENTIFIQUES MEDICALES ELSEVIER, 23 RUE LINOIS, 75724 PARIS, FRANCE.
DT General Review; Journal
LA English
REC Reference Count: 58
ED Entered STN: 3 Feb 2005
Last Updated on STN: 3 Feb 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 4 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Thrombospondin 2 functions as an endogenous regulator of angiogenesis and inflammation in rheumatoid arthritis
AB Thrombospondin 2 (TSP2), a matricellular protein with a primary role in modulating cell-matrix interactions, has been implicated in tissue repair and foreign body responses. Here we show that TSP2 has regulatory function in the chronic inflammatory lesions of rheumatoid arthritis. Tissue TSP2, produced by synovial fibroblasts, endothelial cells, and macrophages correlated not only with the intensity of angiogenesis but also with the architecture of lymphoid infiltrates. Synovial tissues with diffuse inflammatory infiltrates had high levels of TSP2, whereas synovial tissues with ectopic germinal center reactions and T cell-B cell aggregates produced low levels. Cell-based gene therapy with TSP2 was used to examine the in vivo effects of the matrix protein on neoangiogenesis and lymphoid organization. Human synovium-severe combined immunodeficiency (SCID)) mouse chimeras were treated with TSP2-transfected fibroblasts deposited into the peritoneum. Overexpression of TSP2 led to the accumulation of TSP2 protein in the inflamed synovium and resulted in a prompt inhibition of lesional. vascularization. Beside its anti-angiogenic activity, TSP2 also suppressed the production of the proinflammatory mediators, hiterferon-gamma and tumor necrosis factor-alpha, and induced the depletion of tissue-residing T cells. We propose that TSP2 is an endogenous regulator of angiogenesis and autoimmune inflammation in the synovium and represents a protective mechanism preventing ectopic lympho-organogenesis and persistent inflammation in this tissue site.
AN 2004:1067096 SCISEARCH
GA The Genuine Article (R) Number: 874WL
TI Thrombospondin 2 functions as an endogenous regulator of angiogenesis and inflammation in rheumatoid arthritis
AU Park Y W; Kang Y M; Butterfield J; Detmar M; Goronzy J J; Weyand C M (Reprint)
CS Emory Univ, Sch Med, Lowance Ctr Human Immunol, Dept Med, Room 1014, Woodruff Mem Res Bldg, 101 Woodruff Circ, Atlanta, GA 30322 USA (Reprint); Emory Univ, Sch Med, Lowance Ctr Human Immunol, Dept Med, Atlanta, GA 30322 USA; Massachusetts Gen Hosp, Dept Dermatol, Boston, MA 02114 USA; Harvard Univ, Sch Med, Boston, MA USA
cweyand@emory.edu
CYA USA
SO AMERICAN JOURNAL OF PATHOLOGY, (DEC 2004) Vol. 165, No. 6, pp. 2087-2098.
ISSN: 0002-9440.
PB AMER SOC INVESTIGATIVE PATHOLOGY, INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3993 USA.
DT Article; Journal
LA English
REC Reference Count: 56
ED Entered STN: 6 Jan 2005
Last Updated on STN: 6 Jan 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 5 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on

STN
TI Inflammation-responsive promoters for fine-tuned gene therapy in rheumatoid arthritis
AB The inflamed joints of rheumatoid arthritis (RA) patients are ideally suited for gene therapy applications that induce local production of potent anti-inflammatory biologicals. The precise and absolute targeting needed when treating cancer is not necessary in RA. However, the challenge is to regulate transgene expression to meet variable physiological demands during the intermittent course of the disease in RA patients. Thus, a biosensing system with an inducible transcriptional switch that allows robust but adjustable transgene expression is required. Inflammation-inducible promoters are likely candidates to achieve precise control of transgene expression by physiologically driven processes. Acute-phase proteins, pro-inflammatory cytokines, heat-shock proteins and hypoxia-responsive genes are all related to the pathogenesis of RA, and their promoters can be exploited for disease-inducible transgene expression. With this, gene therapy enters a new era, that of temporal control of the therapeutic transgene expression. In addition to the reversible transcriptional switch, the ideal expression system also contains an amplification loop for high transgene expression and a drug-controllable switch to allow intervention by the physician. The merging of these modalities may provide a flexible system to fine-tune transgene expression, which is a prerequisite for the implementation of gene therapy in RA.
AN 2004:977495 SCISEARCH
GA The Genuine Article (R) Number: 865RQ
TI Inflammation-responsive promoters for fine-tuned gene therapy in rheumatoid arthritis
AU van de Loo F A J (Reprint)
CS Univ Nijmegen, Med Ctr, Nijmegen Ctr Mol Life Sci, Dept Rheumatol, Geert Grootepl 26-28, NL-6500 HB Nijmegen, Netherlands (Reprint); Univ Nijmegen, Med Ctr, Nijmegen Ctr Mol Life Sci, Dept Rheumatol, NL-6500 HB Nijmegen, Netherlands
A.vandeloo@reuma.umcn.nl
CYA Netherlands
SO CURRENT OPINION IN MOLECULAR THERAPEUTICS, (OCT 2004) Vol. 6, No. 5, pp. 537-545.
ISSN: 1464-8431.
PB CURRENT DRUGS LTD, MIDDLESEX HOUSE, 34-42 CLEVELAND ST, LONDON W1P 6LB, ENGLAND.
DT Article; Journal
LA English
REC Reference Count: 63
ED Entered STN: 2 Dec 2004
Last Updated on STN: 2 Dec 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 6 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy for autoimmune diseases
AB Autoimmune diseases are threatening an increasing number of patients in developed countries, representing one of the major causes of disability and an enormous social cost. Current therapies mainly treat the symptoms of autoimmune diseases and are only partially able to interfere with disease evolution, and therefore decrease the degree of physical impairment. Thus, the development of new therapeutic strategies is imperative, This review focuses on gene therapy, as one possible alternative approach to the treatment of autoimmune disorders. The potential of gene therapy to specifically target tissues affected by autoimmune aggression, and its ability to interfere with the destructive pathogenic process while providing functional replacement and fostering reparative mechanisms will be emphasized. Gene therapy studies in experimental models of diabetes, rheumatoid arthritis and multiple

sclerosis are reviewed.
AN 2004:977494 SCISEARCH
GA The Genuine Article (R) Number: 865RQ
TI Gene therapy for autoimmune diseases
AU Furlan R (Reprint); Butti E; Pluchino S; Martino G
CS San Raffaele Sci Inst, DIBIT, Neuroimmunol Unit, Via Olgettina 58, I-20132 Milan, Italy (Reprint); San Raffaele Sci Inst, DIBIT, Neuroimmunol Unit, I-20132 Milan, Italy; San Raffaele Sci Inst, Dept Neurol & Neurophysiol, I-20132 Milan, Italy
furlan.roberto@hsr.it
CYA Italy
SO CURRENT OPINION IN MOLECULAR THERAPEUTICS, (OCT 2004) Vol. 6, No. 5, pp. 525-536.
ISSN: 1464-8431.
PB CURRENT DRUGS LTD, MIDDLESEX HOUSE, 34-42 CLEVELAND ST, LONDON W1P 6LB, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 158
ED Entered STN: 2 Dec 2004
Last Updated on STN: 2 Dec 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 11 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy for autoimmune diseases: Quo Vadis?
AB Biological therapies using antibodies and cytokines are becoming widespread for the treatment of chronic inflammatory autoimmune diseases. However, these treatments have several limitations - such as expense, the need for repeated injections and unwanted side-effects - that can be overcome by genetic delivery. This review summarizes the ingenuity, sophistication and variety of gene-therapy approaches that have been taken in the design of therapeutic molecules and vectors, the engineering of cells and the regulation of gene expression for the targeting of disease outcome. We focus our attention on multiple sclerosis, type 1 diabetes and rheumatoid arthritis.
AN 2004:886906 SCISEARCH
GA The Genuine Article (R) Number: 859EK
TI Gene therapy for autoimmune diseases: Quo Vadis?
AU Chernajovsky Y (Reprint); Gould D J; Podhajcer O L
CS Univ London, Barts & London Queen Marys Sch Med & Dent, William Harvey Res Inst, Bone & Joint Res Unit, Charterhouse Sq, London EC1M 6BQ, England (Reprint); Univ London, Barts & London Queen Marys Sch Med & Dent, William Harvey Res Inst, Bone & Joint Res Unit, London EC1M 6BQ, England; Univ Buenos Aires, Fac Exact & Nat Sci, CONICET, Inst Leloir, Buenos Aires, DF, Argentina
y.chernajovsky@qmul.ac.uk
CYA England; Argentina
SO NATURE REVIEWS IMMUNOLOGY, (OCT 2004) Vol. 4, No. 10, pp. 800-811.
ISSN: 1474-1733.
PB NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 138
ED Entered STN: 29 Oct 2004
Last Updated on STN: 29 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 12 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Nonviral gene therapy by electrotransfer of HTNF-alpha soluble receptor-i variants and its application to the treatment of experimental arthritis
AN 2004:878537 SCISEARCH

GA The Genuine Article (R) Number: 857YM
TI Nonviral gene therapy by electrotransfer of HTNF-alpha soluble receptor-i variants and its application to the treatment of experimental arthritis
AU Bloquel C (Reprint); Bessis N; Boissier M C; Scherman D; Bigey P
CS Fac Pharm, CNRS, FRE 2463, INSERM, U266, UPCG, F-75006 Paris, France
CYA France
SO GENE THERAPY, (OCT 2004) Vol. 11, Supp. [1], pp. S124-S125. MA 9. ISSN: 0969-7128.
PB NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND.
DT Conference; Journal
LA English
REC Reference Count: 6
ED Entered STN: 29 Oct 2004
Last Updated on STN: 29 Oct 2004

L10 ANSWER 13 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Gene therapy in animal models of rheumatoid arthritis: are we ready for the patients?
AB Rheumatoid arthritis (RA) is a chronic inflammatory disease of the synovial joints, with progressive destruction of cartilage and bone. Anti-tumour necrosis factor-alpha therapies (e.g. soluble tumour necrosis factor receptors) ameliorate disease in 60-70% of patients with RA. However, the need for repeated systemic administration of relatively high doses in order to achieve constant therapeutic levels in the joints, and the reported side effects are downsides to this systemic approach. Several gene therapeutic approaches have been developed to ameliorate disease in animal models of arthritis either by restoring the cytokine balance or by genetic synovectomy. In this review we summarize strategies to improve transduction of synovial cells, to achieve stable transgene expression using integrating viruses such as adeno-associated viruses, and to achieve transcriptionally regulated expression so that drug release can meet the variable demands imposed by the intermittent course of RA. Evidence from animal models convincingly supports the application of gene therapy in RA, and the feasibility of gene therapy was recently demonstrated in phase I clinical trials.
AN 2004:833051 SCISEARCH
GA The Genuine Article (R) Number: 855RU
TI Gene therapy in animal models of rheumatoid arthritis: are we ready for the patients?
AU van de Loo F A J (Reprint); Smeets R L; van den Berg W B
CS Univ Med Ctr Nijmegen, Nijmegen Ctr Mol Life Sci, Dept Rheumatol, Nijmegen, Netherlands (Reprint)
A.vandeloo@reuma.umcn.nl
CYA Netherlands
SO ARTHRITIS RESEARCH & THERAPY, (2004) Vol. 6, No. 5, pp. 183-196. ISSN: 1478-6362.
PB BIOMED CENTRAL LTD, MIDDLESEX HOUSE, 34-42 CLEVELAND ST, LONDON W1T 4LB, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 117
ED Entered STN: 15 Oct 2004
Last Updated on STN: 15 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 15 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Recent developments in molecular therapeutic approaches for rheumatoid arthritis
AB Rheumatoid arthritis is a debilitating systemic autoimmune disease characterized by chronic synovial inflammation, which

results in the progressive destruction of diseased joints. Advances in understanding the disease pathogenesis have led to the clinical introduction of biological inhibitors of inflammation or articular destruction. However, frequency of administration, cost and systemic side effects have driven efforts to develop gene therapeutic transfer strategies. This article reviews recent progress in the application of viral and non-viral vectors to target therapeutic genes for in vivo delivery.

AN 2004:817648 SCISEARCH
GA The Genuine Article (R) Number: 852KM
TI Recent developments in molecular therapeutic approaches for rheumatoid arthritis
AU Woods A; Hobson P; Klavinskis L S (Reprint)
CS Univ London Kings Coll, Guys Kings & St Thomas Sch Med, Peter Gorer Dept Immunobiol, St Thomas St, London SE1 9RT, England (Reprint); Univ London Kings Coll, Guys Kings & St Thomas Sch Med, Peter Gorer Dept Immunobiol, London SE1 9RT, England
linda.klavinskis@kci.ac.uk
CYA England
SO CURRENT OPINION IN MOLECULAR THERAPEUTICS, (AUG 2004) Vol. 6, No. 4, pp. 395-402.
ISSN: 1464-8431.
PB CURRENT DRUGS LTD, MIDDLESEX HOUSE, 34-42 CLEVELAND ST, LONDON W1P 6LB, ENGLAND.
DT Article; Journal
LA English
REC Reference Count: 75
ED Entered STN: 8 Oct 2004
Last Updated on STN: 8 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 19 OF 638 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Targeted gene therapy: frontiers in the development of 'smart drugs'
AB Chronic diseases, particularly malignancies and immune-mediated inflammatory diseases (IMIDs), are a challenging frontier for clinical diagnosis and treatment, as well as for biomedical research. Current treatment regimens are frequently insufficient and thus new treatment strategies are needed. Novel therapies for disabling such diseases should provide improvements with respect to safety, efficacy and cost. To fulfill these three key criteria, recent research efforts have focused on the development of 'smart drugs'. This review highlights some examples of the rapidly expanding possibilities that current biotechnology has to offer in the development of novel therapeutic strategies for complex diseases such as IMIDs. Special attention is given to advances in, and limitations of, controlled and targeted gene product application in inflammatory diseases.
AN 2004:606187 SCISEARCH
GA The Genuine Article (R) Number: 832XM
TI Targeted gene therapy: frontiers in the development of 'smart drugs'
AU Tarner I H (Reprint); Muller-Ladner U; Fathman C G
CS Univ Hosp Regensburg, Dept Internal Med 1, D-93042 Regensburg, Germany (Reprint); Stanford Univ, Sch Med, Dept Med, Div Rheumatol & Immunol, Stanford, CA 94305 USA
cfathman@stanford.edu
CYA Germany; USA
SO TRENDS IN BIOTECHNOLOGY, (JUN 2004) Vol. 22, No. 6, pp. 304-310.
ISSN: 0167-7799.
PB ELSEVIER SCIENCE LONDON, 84 THEOBALDS RD, LONDON WC1X 8RR, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 87
ED Entered STN: 23 Jul 2004

Last Updated on STN: 23 Jul 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

=> s l10 nad clitocine
MISSING OPERATOR L10 NAD
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l10 and clitocine
L11 0 L10 AND CLITOCINE

=> s (gene(w)therapy) and (kidney(w)stones)
L12 4 (GENE(W) THERAPY) AND (KIDNEY(W) STONES)

=> d l12 1-4 ti

L12 ANSWER 1 OF 4 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Hyperoxaluria. and systemic oxalosis: current therapy and future directions

L12 ANSWER 2 OF 4 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Molecular etiology of primary hyperoxaluria type 1: New directions for treatment

L12 ANSWER 3 OF 4 MEDLINE on STN
TI Hyperoxaluria and systemic oxalosis: current therapy and future directions.

L12 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2006 ACS on STN
TI Hyperoxaluria and systemic oxalosis: current therapy and future directions

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 2 DUP REM L12 (2 DUPLICATES REMOVED)

=> d l13 1-2 ti

L13 ANSWER 1 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 1
TI Hyperoxaluria. and systemic oxalosis: current therapy and future directions

L13 ANSWER 2 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Molecular etiology of primary hyperoxaluria type 1: New directions for treatment

=> d l13 1-2 ti abs bib

L13 ANSWER 1 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 1
TI Hyperoxaluria. and systemic oxalosis: current therapy and future directions
AB Excessive urinary oxalate excretion, termed hyperoxaluria, may arise from inherited or acquired diseases. The most severe forms are caused by increased endogenous production of oxalate related to one of several inborn errors of metabolism, termed primary hyperoxaluria. Recurrent kidney stones and progressive medullary nephrocalcinosis lead to the loss of kidney function, requiring dialysis or transplantation, accompanied by systemic oxalate deposition that is termed

systemic oxalosis. For most primary hyperoxalurias, accurate diagnosis leads to the use of therapies that include pyridoxine supplementation, urinary crystallisation inhibitors, hydration with enteral fluids and, in the near future, probiotic supplementation or other innovative therapies. These therapies have varying degrees of. success, and none represent a cure. Organ transplantation results in reduced patient and organ survival when compared with national statistics. Exciting new approaches under investigation include the restoration of defective enzymatic activity through the use of chemical chaperones and hepatocyte cell transplantation, or recombinant gene therapy for enzyme replacement. Such approaches give hope for a future therapeutic cure for primary hyperoxaluria that includes correction of the underlying genetic defect without exposure to the life-long dangers associated with organ transplantation.

AN 2006:966888 SCISEARCH
GA The Genuine Article (R) Number: 091GN
TI Hyperoxaluria. and systemic oxalosis: current therapy and future directions
AU Bobrowski A E; Langman C B (Reprint)
CS Northwestern Univ, Childrens Mem Hosp, Feinberg Sch Med, Div Kidney Dis, Dept Pediat, 2300 Childrens Plaza 37, Chicago, IL 60614 USA (Reprint); Northwestern Univ, Childrens Mem Hosp, Feinberg Sch Med, Div Kidney Dis, Dept Pediat, Chicago, IL 60614 USA
abobrowski@childrensmemorial.org; c-langman@northwestern.edu
CYA USA
SO EXPERT OPINION ON PHARMACOTHERAPY, (OCT 2006) Vol. 7, No. 14, pp. 1887-1896.
ISSN: 1465-6566.
PB INFORMA HEALTHCARE, TELEPHONE HOUSE, 69-77 PAUL STREET, LONDON EC2A 4LQ, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 60
ED Entered STN: 20 Oct 2006
Last Updated on STN: 20 Oct 2006
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L13 ANSWER 2 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Molecular etiology of primary hyperoxaluria type 1: New directions for treatment
AB Primary hyperoxaluria type 1 (PH1) is a rare autosomal-recessive disorder caused by a deficiency of the liver-specific enzyme alanine: glyoxylate aminotransferase (AGT). AGT deficiency results in increased synthesis and excretion of the metabolic end-product oxalate and deposition of insoluble calcium oxalate in the kidney and urinary tract. Classic treatments for PH1 have tended to address the more distal aspects of the disease process (i.e. the symptoms rather than the causes). However, advances in the understanding of the molecular etiology of PH1 over the past decade have shifted attention towards the more proximal aspects of the disease process (i.e. the causes rather than the symptoms). The determination of the crystal structure of AGT has enabled the effects of some of the most important missense mutations in the AGXT gene to be rationalised in terms of AGT folding, dimerization and stability. This has opened up new possibilities for the design pharmacological agents that might counteract the destabilizing effects of these mutations and which might be of use for the treatment of a potentially life-threatening and difficult-to-treat disease. Copyright (C) 2005 S. Karger AG, Basel.
AN 2005:705908 SCISEARCH
GA The Genuine Article (R) Number: 942TQ
TI Molecular etiology of primary hyperoxaluria type 1: New directions for treatment
AU Danpure C J (Reprint)
CS Univ Coll London, Dept Biol, Gower St, London WC1E 6BT, England (Reprint); Univ Coll London, Dept Biol, London WC1E 6BT, England

c.danpure@ucl.ac.uk
CYA England
SO AMERICAN JOURNAL OF NEPHROLOGY, (2005) Vol. 25, No. 3, pp. 303-310.
ISSN: 0250-8095.
PB KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
DT General Review; Journal
LA English
REC Reference Count: 53
ED Entered STN: 22 Jul 2005
Last Updated on STN: 22 Jul 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

=> s (gene(w)therapy) and (graft-versus-host)
L14 206 (GENE(W) THERAPY) AND (GRAFT-VERSUS-HOST)

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15 134 DUP REM L14 (72 DUPLICATES REMOVED)

=> d l15 1-20 ti

L15 ANSWER 1 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 1
TI Analysis of transgene-specific immune responses that limit the in vivo persistence of adoptively transferred HSV-TK-modified donor T cells after allogeneic hematopoietic cell transplantation

L15 ANSWER 2 OF 134 MEDLINE on STN DUPLICATE 2
TI Allogeneic MHC gene transfer enhances antitumor activity of allogeneic hematopoietic stem cell transplantation without exacerbating graft -versus-host disease.

L15 ANSWER 3 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 3
TI Suicide gene therapy of graft-versus -host disease induced by central memory human T lymphocytes

L15 ANSWER 4 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Retroviral vector integration deregulates gene expression but has no consequence on the biology and function of transplanted T cells

L15 ANSWER 5 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 4
TI Initial depletion of regulatory T cells: the missing solution to preserve the immune functions of T lymphocytes designed for cell therapy

L15 ANSWER 6 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 5
TI Key factors in experimental mouse hematopoletic stem cell transplantation

L15 ANSWER 7 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 6
TI Retroviral vector insertions in T-lymphocytes used for suicide gene therapy occur in gene groups with specific molecular functions

L15 ANSWER 8 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Cutaneous graft versus host disease-like histopathological features following gene therapy

L15 ANSWER 9 OF 134 MEDLINE on STN DUPLICATE 7
TI TRAIL-transduced dendritic cells protect mice from acute graft-

versus-host disease and leukemia relapse.

L15 ANSWER 10 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Suicide gene therapy of graft-versus -host disease induced by central memory human T lymphocytes.

L15 ANSWER 11 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 8
TI Development of an inducible suicide gene system based on human caspase 8

L15 ANSWER 12 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Cytokine-mediated signalling and early defects in lymphoid development

L15 ANSWER 13 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Differential gene expression profiling of CD34(+) CD133(+) umbilical cord blood hematopoietic stem progenitor cells

L15 ANSWER 14 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 9
TI T cell suicide gene therapy to aid haematopoietic stem cell transplantation

L15 ANSWER 15 OF 134 CAPLUS COPYRIGHT 2006 ACS on STN
TI Research advance in preventive therapy of graft versus host disease

L15 ANSWER 16 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Suicide gene therapy for human T cell mediated graft versus host disease in a murine xenograft model

L15 ANSWER 17 OF 134 CAPLUS COPYRIGHT 2006 ACS on STN
TI CD8 α chain polypeptides for inhibiting alloantigen-specific immune responses and for treating allotransplant rejection

L15 ANSWER 18 OF 134 CAPLUS COPYRIGHT 2006 ACS on STN
TI Modulators and antagonists of Notch signalling for reducing risk of graft versus host disease and preventing infection

L15 ANSWER 19 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 10
TI Kinetics of in vivo elimination of suicide gene-expressing T cells affects engraftment, graft-versus-host disease, and graft-versus-leukemia after allogeneic bone marrow transplantation

L15 ANSWER 20 OF 134 MEDLINE on STN
TI Preferential retroviral-mediated transduction of EBV- and CMV-specific T cells after polyclonal T-cell activation.

=> d l15 14 15 16 19 ti abs bib

L15 ANSWER 14 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 9
TI T cell suicide gene therapy to aid haematopoietic stem cell transplantation
AB Graft versus host disease (GVHD) is a T cell mediated phenomenon that arises following allogeneic haematopoietic stem cell transplantation, and may be particularly severe in the context of human leukocyte antigen (HLA) mismatched procedures. Although GVHD can

be largely abrogated through T cell depletion, such measures result in loss of graft potency and reduced anti-viral and anti-leukaemic effects. The genetic modification of T cells to carry a suicide gene mechanism has been advocated as means of allowing T cells to be harnessed for their beneficial effects, and safely eliminated in the event of significant GVHD.

The feasibility of the strategy has been demonstrated in clinical studies using T cells modified by retroviral transduction to encode the herpes simplex thymidine kinase (HSVTK) gene to treat patients with haematological malignancies. However, a number of limitations associated with current protocols have become apparent. Most notably, the process of retroviral transduction, which requires pre-activation of T cells, appears to impair subsequent functional potential. Efforts are now directed towards circumventing the pre-activation requirements of retroviral vectors by using alternative lentiviral systems, in association with improved suicide gene/prodrug combinations.

AN 2005:164498 SCISEARCH
GA The Genuine Article (R) Number: 893KQ
TI T cell suicide gene therapy to aid haematopoietic stem cell transplantation
AU Qasim W (Reprint); Gaspar H B; Thrasher A J
CS Inst Child Hlth, Mol Immunol Unit, 30 Guilford St, London WC1N 1EH, England (Reprint); Inst Child Hlth, Mol Immunol Unit, London WC1N 1EH, England
W.Qasim@ich.ucl.ac.uk
CYA England
SO CURRENT GENE THERAPY, (FEB 2005) Vol. 5, No. 1, pp. 121-132.
ISSN: 1566-5232.
PB BENTHAM SCIENCE PUBL LTD, EXECUTIVE STE Y26, PO BOX 7917, SAIF ZONE, 1200 BR SHARJAH, U ARAB EMIRATES.
DT General Review; Journal
LA English
REC Reference Count: 69
ED Entered STN: 24 Feb 2005
Last Updated on STN: 24 Feb 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L15 ANSWER 15 OF 134 CAPLUS COPYRIGHT 2006 ACS on STN
TI Research advance in preventive therapy of graft versus host disease
AB A review. Graft vs. host disease (GVHD) is a problem in transplantation of allogene hematopoietic stem cell. Its immune response is related with interaction of T cells, natural kill cells (NK) from donators and specific cells from receptors. It is induced by cytokines, lymphocytes and target cells, and results in injury of multiple organs. Monoclonal antibody, gene therapy, cell therapy and vaccine therapy can selectively remove cells which induce GVHD, establish xenogenous specific transplantation tolerance, enhance survival rate and ameliorate quality of survival. Transplantation of hematopoietic stem cell is studied in the fields of these biol. methods.
AN 2005:1157080 CAPLUS
DN 144:252211
TI Research advance in preventive therapy of graft versus host disease
AU Yang, Fan
CS Affiliated Hospital, Academy of Military Medical Sciences, Beijing, 100039, Peop. Rep. China
SO Baixuebing Linbaliu (2005), 14(1), 59-61
CODEN: BLAIBD; ISSN: 1009-9921
PB Baixuebing Linbaliu Zazhi Bianjibu
DT Journal; General Review
LA Chinese

L15 ANSWER 16 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Suicide gene therapy for human T cell mediated graft versus host disease in a murine xenograft model
AN 2005:368455 SCISEARCH
GA The Genuine Article (R) Number: 902CM
TI Suicide gene therapy for human T cell mediated graft versus host disease in a murine xenograft model
AU Nervi B (Reprint); Rettig M P; Ritchey J; Walker J; Bauer G; Herrbrich P E; Bonyhadi M L; Nolta J A; DiPersio J F
CS Washington Univ, Sch Med, St Louis, MO USA; Xcyte Therapies Inc, Seattle, WA USA
CYA USA
SO BIOLOGY OF BLOOD AND MARROW TRANSPLANTATION, (FEB 2005) Vol. 11, No. 2, Supp. [1], pp. 45-46. MA 134.
ISSN: 1083-8791.
PB CARDEN JENNINGS PUBL CO LTD, BLAKE CTR, STE 200, 1224 W MAIN ST, CHARLOTTESVILLE, VA 22903 USA.
DT Conference; Journal
LA English
REC Reference Count: 0
ED Entered STN: 14 Apr 2005
Last Updated on STN: 14 Apr 2005

L15 ANSWER 19 OF 134 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 10
TI Kinetics of in vivo elimination of suicide gene-expressing T cells affects engraftment, graft-versus-host disease, and graft-versus-leukemia after allogeneic bone marrow transplantation
AB Suicide gene therapy is one approach being evaluated for the control of graft-vs-host disease (GVHD) after allogeneic bone marrow transplantation (BMT). We recently constructed a novel chimeric suicide gene in which the entire coding region of HSV thymidine kinase (HSV-tk) was fused in-frame to the extracellular and transmembrane domains of human CD34 (DeltaCD34-tk). DeltaCD34-tk is an attractive candidate as a suicide gene in man because of the ensured expression of HSV-tk in all selected cells and the ability to rapidly and efficiently purify gene-modified cells using clinically approved CD34 immunoselection techniques. In this study we assessed the efficacy of the DeltaCD34-tk suicide gene in the absence of extended ex vivo manipulation by generating transgenic animals that express DeltaCD34-tk in the peripheral and thymic T cell compartments using the CD2 locus control region. We found that DeltaCD34-tk-expressing T cells could be purified to near homogeneity by CD34 immunoselection and selectively eliminated ex vivo and in vivo when exposed to low concentrations of GCV. The optimal time to administer GCV after allogeneic BMT with DeltaCD34-tk-expressing transgenic T cells was dependent on the intensity of the conditioning regimen, the leukemic status of the recipient, and the dose and timing of T cell infusion. Importantly, we used a controlled graft-vs-host reaction to promote alloengraftment in sublethally irradiated mice and provide a graft-vs-leukemia effect in recipients administered a delayed infusion of DeltaCD34-tk-expressing T cells. This murine model demonstrates the potential usefulness of DeltaCD34-tk-expressing T cells to control GVHD, promote alloengraftment, and provide a graft-vs-leukemia effect in man.
AN 2004:863643 SCISEARCH
GA The Genuine Article (R) Number: 854CN
TI Kinetics of in vivo elimination of suicide gene-expressing T cells affects engraftment, graft-versus-host disease, and graft-versus-leukemia after allogeneic bone marrow transplantation
AU Rettig M P; Ritchey J K; Prior J L; Haug J S; Piwnica-Worms D; DiPersio J F (Reprint)
CS Washington Univ, Sch Med, Div Oncogen, Siteman Canc Ctr, Box 8007, 660 S Euclid Ave, St Louis, MO 63110 USA (Reprint); Washington Univ, Sch Med, Div Oncogen, Siteman Canc Ctr, St Louis, MO 63110 USA; Washington Univ, Sch Med, Mallinckrodt Inst Radiol, Mol Imaging Ctr, St Louis, MO 63110

USA; Washington Univ, Sch Med, Dept Mol Biol & Pharmacol, St Louis, MO 63110 USA
jdipersi@im.wustl.edu
CYA USA
SO JOURNAL OF IMMUNOLOGY, (15 SEP 2004) Vol. 173, No. 6, pp. 3620-3630. ISSN: 0022-1767.
PB AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DT Article; Journal
LA English
REC Reference Count: 54
ED Entered STN: 22 Oct 2004
Last Updated on STN: 22 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

=> s l15 and (adenyl?(w)cyclase)
L16 0 L15 AND (ADENYL?(W) CYCLASE)

=> s l15 and (nonsense)
L17 0 L15 AND (NONSENSE)

=> s l15 and (mutation)
L18 2 L15 AND (MUTATION)

=> d l18 1-2 ti

L18 ANSWER 1 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Cytokine-mediated signalling and early defects in lymphoid development

L18 ANSWER 2 OF 2 MEDLINE on STN
TI T cell transduction and suicide with an enhanced mutant thymidine kinase.

=> d l18 1-2 ti abs bib

L18 ANSWER 1 OF 2 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Cytokine-mediated signalling and early defects in lymphoid development
AB Purpose of review
The aim of the review is to report on recent advances in cytokine-mediated signalling, as illustrated by the study of natural human mutants. In particular, the role of cytokines and cytokine-mediated signalling in human T-cell development is analysed in detail, and currently available forms of treatment including experimental trials are described.
Recent findings
Defects of the cytokine/JAK/STAT axis have been recently described as responsible for human Severe Combined Immune Deficiency. In particular, defects in gamma c, JAK3 and IL7RA have been analysed in terms of development of novel diagnostic tools as well as of new therapeutic agents for the treatment of autoimmune diseases and graft-versus-host disease.
Summary
Dissection of the genetic defects underlying the various forms of Severe Combined Immune Deficiency has helped develop new and more accurate diagnostic assays and novel forms of treatment.
AN 2005:1217017 SCISEARCH
GA The Genuine Article (R) Number: 988GY
TI Cytokine-mediated signalling and early defects in lymphoid development
AU Giliani S (Reprint); Mella P; Savoldi G; Mazzolari E
CS Univ Brescia, Spedali Civili, Angelo Nocivelli Inst Mol Med, Pzzale Spedali Civili 1, I-25123 Brescia, Italy (Reprint); Univ Brescia, Spedali Civili, Angelo Nocivelli Inst Mol Med, I-25123 Brescia, Italy; Univ Brescia, Dept Pediat, I-25123 Brescia, Italy

giliani@master.cci.unibs.it
CYA Italy
SO CURRENT OPINION IN ALLERGY AND CLINICAL IMMUNOLOGY, (DEC 2005) Vol. 5, No. 6, pp. 519-524.
ISSN: 1528-4050.
PB LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3261 USA.
DT General Review; Journal
LA English
REC Reference Count: 44
ED Entered STN: 15 Dec 2005
Last Updated on STN: 15 Dec 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L18 ANSWER 2 OF 2 MEDLINE on STN
TI T cell transduction and suicide with an enhanced mutant thymidine kinase.
AB Retroviral transfer of Herpes simplex virus thymidine kinase to T cells has been used to confer sensitivity to the antiviral agent ganciclovir. This has allowed therapeutic approaches to be developed in which T cells mediating graft-versus-host disease after bone marrow transplantation can be selectively eliminated by the administration of ganciclovir. Although the strategy has been shown to be generally successful in early clinical trials, there are concerns about possible resistance to ganciclovir and the risk of myelosuppressive side-effects at the doses required to induce T cell suicide. We have incorporated the enhanced mutant HSV-TKSR39 into retroviral vectors tailored to exhibit high levels of expression in T cells and have used protocols optimized for the transduction and selection of primary lymphocytes. We demonstrate that leukemic and primary T cells can be efficiently transduced and highly enriched under conditions that should be readily adaptable for clinical use. T cells carrying HSV-TKSR39 were inhibited by exposure to ganciclovir at concentrations an order of magnitude below those required for wild-type HSV-TK. The less toxic agent aciclovir also eliminated T cells transduced with HSV-TKSR39 (but not HSV-TK), underlining the increased therapeutic potential of the mutant suicide gene system in the bone marrow transplantation setting.
AN 2002298885 MEDLINE
DN PubMed ID: 12040465
TI T cell transduction and suicide with an enhanced mutant thymidine kinase.
AU Qasim W; Thrasher A J; Buddle J; Kinnon C; Black M E; Gaspar H B
CS Molecular Immunology Unit, Institute of Child Health, University College London, UK.
SO Gene therapy, (2002 Jun) Vol. 9, No. 12, pp. 824-7.
Journal code: 9421525. ISSN: 0969-7128.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200208
ED Entered STN: 2 Jun 2002
Last Updated on STN: 10 Aug 2002
Entered Medline: 9 Aug 2002

=> s (gene(w)therapy) and (Alzheim? or parkinson? or neurodegen?)
L19 3159 (GENE(W) THERAPY) AND (ALZHEIM? OR PARKINSON? OR NEURODEGEN?)

=> s l19 and (adenyl?(w)cclase)
L20 0 L19 AND (ADENYL?(W) CCLASE)

=> s l19 and (adenyl?(w)cyclase)
L21 8 L19 AND (ADENYL?(W) CYCLASE)

=> dup rem l21
PROCESSING COMPLETED FOR L21

L22 8 DUP REM L21 (0 DUPLICATES REMOVED)

=> d l22 1-8 ti

L22 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Methods of stimulating axonal growth of CNS neurons using Nogo receptor antagonists in combination with growth factors

L22 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Neuronal and optic nerve gene expression patterns in the optic nerve axotomy model of neuronal degeneration

L22 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Markers of neuronal cell death and their use in diagnosis and therapy

L22 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Selective target cell activation by expression of a G protein-coupled receptor activated superiorly by synthetic ligand (RASSL) for disease treatment and disease modeling

L22 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Constitutively active, hypersensitive, and nonfunctional receptors as novel therapeutic agents

L22 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses

L22 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses

L22 ANSWER 8 OF 8 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Physiological relevance and functional potential of central nervous system-derived cell lines

=> d l22 2 3 4 6 7 ti abs bib

L22 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Neuronal and optic nerve gene expression patterns in the optic nerve axotomy model of neuronal degeneration
AB The optic nerve axotomy model of optic nerve degeneration in mouse exhibits rapid changes in gene expression. Genes identified by microarray anal. as differentially expressed or modulated in this model, can be used diagnostically, therapeutically, and in drug discovery. These results provide clues to underlying mol. processes occurring during optic nerve degeneration, and provide direction for future cell-based studies.
AN 2004:60635 CAPLUS
DN 140:109564
TI Neuronal and optic nerve gene expression patterns in the optic nerve axotomy model of neuronal degeneration
IN Zack, Donald J.; Quigley, Harry A.
PA The Johns Hopkins University, USA
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004007675	A2	20040122	WO 2003-US21738	20030714

WO 2004007675 A3 20040819
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2003253883 A1 20040202 AU 2003-253883 20030714
US 2004081652 A1 20040429 US 2003-617888 20030714
PRAI US 2002-395821P P 20020715
WO 2003-US21738 W 20030714

L22 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Markers of neuronal cell death and their use in diagnosis and therapy
AB Neuronal cell death, as modeled by removal of serum or NGF from growth medium, is characterized by many changes in gene expression. Gene expression was compared before and after withdrawal of serum or NGF. These results provide clues to underlying mol. processes occurring during neuronal and photoreceptor degeneration, and provide direction for future cell-based studies.
AN 2004:60633 CAPLUS
DN 140:126705
TI Markers of neuronal cell death and their use in diagnosis and therapy
IN Zack, Donald J.; Kageyama, Masaaki
PA The Johns Hopkins University, USA
SO PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004007673	A2	20040122	WO 2003-US21729	20030714
WO 2004007673	A3	20041118		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003249054	A1	20040202	AU 2003-249054	20030714
US 2004086511	A1	20040506	US 2003-617885	20030714
PRAI US 2002-395753P	P	20020712		
WO 2003-US21729	W	20030714		

L22 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Selective target cell activation by expression of a G protein-coupled receptor activated superiorly by synthetic ligand (RASSL) for disease treatment and disease modeling
AB The invention provides a method for selectively activating a target cell, where the target cell expresses a receptor activated superiorly by a synthetic ligand (RASSL) having decreased binding affinity for a selected natural ligand and normal or near normal binding affinity for a synthetic small mol. agonist. Thus, RASSL-mediated activation of target cells does not occur to a significant extent in the presence of natural G protein-coupled receptor ligand, but is significantly stimulated upon exposure to a synthetic small mol. RASSL-expressing target cells are

selectively activated by exposing of the cells to an appropriate synthetic small mol., which in turn binds the RASSL, resulting in G protein activation and triggering of a specific cellular response associated with G protein activation (e.g., cellular proliferation or cellular secretion).

AN 2003:696602 CAPLUS
DN 139:207808
TI Selective target cell activation by expression of a G protein-coupled receptor activated superiorly by synthetic ligand (RASSL) for disease treatment and disease modeling
IN Conklin, Bruce R.
PA USA
SO U.S. Pat. Appl. Publ., 61 pp., Cont.-in-part of U.S. 6,518,480.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2003167476	A1	20030904	US 2002-318661	20021212
	US 6518480	B1	20030211	US 1999-341446	19991220
PRAI	US 1996-622348	B2	19960326		
	US 1999-341446	A2	19991220		
	WO 1997-US5334	W	19970325		

L22 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses
AB The cDNA sequence and the corresponding deduced amino acid sequence of a G-protein receptor putatively identified as a pituitary adenylate cyclase-activating polypeptide (PACAP) receptor are provided. The cDNA was discovered in a cDNA library derived from human cerebellum tissue. Is is structurally related to the G protein-coupled receptor family. It contains an open reading frame encoding a protein of 884 amino acid residues. The protein exhibits the highest degree of homol. to rat PACAP-like receptor. Recombinant techniques for expression of the receptor are described, including (1) expression in COS-7 cells using the pcDNAI/Amp vector, (2) cloning and expression using the baculovirus expression system with the pA2 vector (a modification of the pVL941 vector) in Sf9 cells, and (3) expression via gene therapy with the pMV-7 vector based on the Moloney murine sarcoma virus backbone. Also disclosed are methods for utilizing such polypeptides for identifying antagonists and agonists to such polypeptides. Antagonists against such polypeptides may be used therapeutically to treat PACAP hypersecretory conditions and to create pharmacol. amnesia models, while the agonists may be employed to treat amnesia and Alzheimer's disease. Also disclosed are diagnostic methods for detecting a mutation in the PACAP receptor nucleic acid sequences and detecting a level of the soluble form of the receptors in a sample derived from a host.
AN 1998:398410 CAPLUS
DN 129:64073
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses
IN Soppet, Daniel R.; Rosen, Craig A.; Ruben, Steven M.; Li, Yi
PA Human Genome Sciences, Inc., USA; Soppet, Daniel R.; Rosen, Craig A.; Ruben, Steven M.; Li, Yi
SO PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 9824900 A1 19980611 WO 1997-US20547 19971121
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
CA 2273198 AA 19980611 CA 1997-2273198 19971121
AU 9876253 A1 19980629 AU 1998-76253 19971121
EP 941327 A1 19990915 EP 1997-949398 19971121
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2001505433 T2 20010424 JP 1998-525590 19971121
PRAI US 1996-32186P P 19961202
WO 1997-US20547 W 19971121
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses
AB The cDNA sequence and the corresponding deduced amino acid sequence of a G-protein receptor putatively identified as a pituitary adenylate cyclase-activating polypeptide (PACAP) receptor are provided. The cDNA was discovered in a cDNA library derived from human cerebellum tissue. Is is structurally related to the G protein-coupled receptor family. It contains an open reading frame encoding a protein of 874 amino acid residues. The protein exhibits the highest degree of homol. to rat PACAP-like receptor with 22.910% identity and 48.607% similarity. Recombinant techniques for expression of the receptor are described, including (1) expression in COS-7 cells using the pcDNAI/Amp vector, (2) cloning and expression using the baculovirus expression system with the pA2 vector (a modification of the pVL941 vector) in Sf9 cells, and (3) expression via gene therapy with the pMV-7 vector based on the Moloney murine sarcoma virus backbone. Also disclosed are methods for utilizing such polypeptides for identifying antagonists and agonists to such polypeptides. Antagonists against such polypeptides may be used therapeutically to treat PACAP hypersecretory conditions and to create pharmacol. amnesia models, while the agonists may be employed to treat amnesia and Alzheimer's disease. Also disclosed are diagnostic methods for detecting a mutation in the PACAP receptor nucleic acid sequences and detecting a level of the soluble form of the receptors in a sample derived from a host.
AN 1997:97257 CAPLUS
DN 126:100279
TI Human G protein-coupled pituitary adenylate cyclase activating polypeptide-like receptor HCEGH45, cloning of its cDNA sequence, and its diagnostic and therapeutic uses
IN Soppet, Daniel R.; Li, Yi; Rosen, Craig A.; Ruben, Steven M.
PA Human Genome Sciences, Inc., USA; Soppet, Daniel R.; Li, Yi; Rosen, Craig A.; Ruben, Steven M.
SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9639439	A1	19961212	WO 1995-US7188	19950606

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN

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        RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT,
            LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE,
            SN, TD, TG
    CA 2221637          AA    19961212    CA 1995-2221637        19950606
    AU 9526634          A1    19961224    AU 1995-26634          19950606
    EP 835264           A1    19980415    EP 1995-921615         19950606
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE
PRAI WO 1995-US7188     A     19950606


=> s (gene(w)therapy) and (cirrhosis)
L23        448 (GENE(W) THERAPY) AND (CIRRHOSIS)

=> s l23 not py>2004
L24        316 L23 NOT PY>2004

=> s l24 and (adenyl?(w)cclase)
L25          0 L24 AND (ADENYL?(W) CCLASE)

=> s l24 and (nonsense)
L26          0 L24 AND (NONSENSE)

=> dup rem l26
L26 HAS NO ANSWERS

=> dup rem l24
PROCESSING COMPLETED FOR L24
L27         224 DUP REM L24 (92 DUPLICATES REMOVED)

=> d l27 1-20 ti

L27  ANSWER 1 OF 224     MEDLINE on STN
TI   A cut above the rest? MMP-8 and liver fibrosis gene
     therapy.

L27  ANSWER 2 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Development of vectors based on SV40 virus: Production, biodistribution
     and applications in the treatment of liver cirrhosis and colon
     cancer

L27  ANSWER 3 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Method for assessing a patient's risk of development or progression of
     liver cirrhosis by genotyping coagulation factors

L27  ANSWER 4 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Antisense nucleic acid sequences and methods for use in the therapeutic
     and preventative treatment, study, diagnosis and prognosis of liver
     related disease inflammatory disease

L27  ANSWER 5 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Adhesion molecules identified from E. coli and human by mining of sequence
     database, and therapeutic and diagnostic use applications

L27  ANSWER 6 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Methods for treating an autoimmune disease using a soluble CTLA4 molecule
     in combination with a DMARD or NSAID

L27  ANSWER 7 OF 224     MEDLINE on STN                  DUPLICATE 1
TI   Alternative approaches for efficient inhibition of hepatitis C virus RNA
     replication by small interfering RNAs.

L27  ANSWER 8 OF 224  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Influence of mutations in the hepatitis B virus genome on virus
     replication and drug resistance - implications for novel antiviral
     strategies
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L27 ANSWER 9 OF 224 MEDLINE on STN DUPLICATE 2
TI Simultaneous transfer of vascular endothelial growth factor and hepatocyte growth factor genes effectively promotes liver regeneration after hepatectomy in cirrhotic rats.

L27 ANSWER 10 OF 224 MEDLINE on STN
TI Blockage of transforming growth factor beta receptors prevents progression of pig serum-induced rat liver fibrosis.

L27 ANSWER 11 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 3
TI Liver transplantation: Challenge of medical necessity and allocation

L27 ANSWER 12 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 4
TI A cut above the rest? - MMP-8 and liver fibrosis gene therapy

L27 ANSWER 13 OF 224 MEDLINE on STN
TI Inhibitory effect of retroviral vector containing anti-sense Smad4 gene on Ito cell line, LI90.

L27 ANSWER 14 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 5
TI Metallothionein gene therapy for chemical-induced liver fibrosis in mice

L27 ANSWER 15 OF 224 MEDLINE on STN DUPLICATE 6
TI Treatment with human metalloproteinase-8 gene delivery ameliorates experimental rat liver cirrhosis.

L27 ANSWER 16 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Induction of cell death in activated hepatic stellate cells by targeted gene expression of the thymidine kinase/ganciclovir system

L27 ANSWER 17 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 7
TI Gene therapy of liver diseases

L27 ANSWER 18 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 8
TI Inhibition of hepatitis C virus NS3-mediated cell transformation by recombinant intracellular antibodies

L27 ANSWER 19 OF 224 CAPLUS COPYRIGHT 2006 ACS on STN
TI Suppression of transforming growth factor-β results in upregulation of transcription of regeneration factors after chronic liver injury

L27 ANSWER 20 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 9
TI Severe pulmonary pathology after intravenous administration of adenovirus vectors in cirrhotic rats

=> d l27 2 3 12 17 ti abs bib

L27 ANSWER 2 OF 224 CAPLUS COPYRIGHT 2006 ACS on STN
TI Development of vectors based on SV40 virus: Production, biodistribution and applications in the treatment of liver cirrhosis and colon cancer
AB Unavailable
AN 2005:1125010 CAPLUS
DN 144:185523

TI Development of vectors based on SV40 virus: Production, biodistribution and applications in the treatment of liver cirrhosis and colon cancer
AU Vera Ugalde, Maria
CS Universidad de Navarra, Pamplona, Spain
SO (2004) 231 pp. Avail.: From degree-granting institution From: Diss. Abstr. Int., C 2005, 66(1), 98
DT Dissertation
LA Spanish

L27 ANSWER 3 OF 224 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method for assessing a patient's risk of development or progression of liver cirrhosis by genotyping coagulation factors
AB A method for determining a patient's risk of rapid fibrosis and/or cirrhosis as a result of HCV infection and/or risk of poor response to therapy comprising the steps of providing a Bayesian model identifying factors contributing to the risk and their relative importance, obtaining information on at least one such factor in relation to the patient and calculating the patient's risk using the model. The factors may include gender, age at infection, viral genotype, Factor V Leiden genotype, P-selectin genotype, alpha-adducin genotype and CETP genotype. Patients with Factor V Leiden (Arg560Gln) may be at a higher risk of developing liver cirrhosis or fibrosis and/or of rapid progression of liver cirrhosis or fibrosis. In particular, disclosed are evidence and confirmation that the factor V Leiden mutation leads to an increased rate of fibrosis in HCV infection. The functional significance of factor V Leiden is well described in that this mutation confers resistance to activated protein C which normally degrades factor V. Increased activity of factor V leads to increased thrombin activity and hence fibrin production Also presented is the hypothesis that those with the polymorphism have a procoagulant state in response to the liver inflammation resulting from HCV which gives rise to increased thrombin (a stellate cell mitogen) generation and increased fibrin deposition. Increased thrombin levels affect stellate cell activation and hence may enhance fibrosis deposition.
AN 2004:824114 CAPLUS
DN 141:275708
TI Method for assessing a patient's risk of development or progression of liver cirrhosis by genotyping coagulation factors
IN Wright, Mark; Thursz, Mark
PA Imperial College Innovations Limited, UK
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004086031	A2	20041007	WO 2004-GB1385	20040326
	WO 2004086031	A3	20041202		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	GB 2003-7076	A	20030327		

L27 ANSWER 12 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 4

TI A cut above the rest? - MMP-8 and liver fibrosis gene therapy
AN 2004:357919 SCISEARCH
GA The Genuine Article (R) Number: 810ER
TI A cut above the rest? - MMP-8 and liver fibrosis gene therapy
AU Iredale J P (Reprint)
CS Univ Southampton, Southampton Gen Hosp, Mail Point 811, Southampton SO16 6YD, Hants, England (Reprint); Univ Southampton, Southampton Gen Hosp, Southampton SO16 6YD, Hants, England
CYA England
SO GASTROENTEROLOGY, (APR 2004) Vol. 126, No. 4, pp. 1199-1201.
ISSN: 0016-5085.
PB W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
DT Editorial; Journal
LA English
REC Reference Count: 19
ED Entered STN: 30 Apr 2004
Last Updated on STN: 30 Apr 2004

L27 ANSWER 17 OF 224 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 7
TI Gene therapy of liver diseases
AB Many liver diseases lack satisfactory treatment and alternative therapeutic options are urgently needed. Gene therapy is a new mode of treatment for both inherited and acquired diseases, based on the transfer of genetic material to the tissues. Genes are incorporated into appropriate vectors in order to facilitate their entrance and function inside the target cells. Gene therapy vectors can be constructed on the basis of viral or non-viral molecular structures. Viral vectors are frequently used, due to their higher transduction efficiency. Both the type of vector and the expression cassette determine the duration, specificity and inducibility of gene expression. A considerable number of preclinical studies indicate that a great variety of liver diseases, including inherited metabolic defects, chronic viral hepatitis, liver cirrhosis and primary and metastatic liver cancer, are amenable to gene therapy. Gene transfer to the liver can also be used to convert this organ into a factory of secreted proteins needed to treat conditions that do not affect the liver itself. Clinical trials of gene therapy for the treatment of inherited diseases and liver cancer have been initiated but human gene therapy is still in its infancy. Recent progress in vector technology and imaging techniques, allowing in vivo assessment of gene expression, will facilitate the development of clinical applications of gene therapy.
AN 2004:676874 SCISEARCH
GA The Genuine Article (R) Number: 83900
TI Gene therapy of liver diseases
AU Prieto J (Reprint); Qian C; Hernandez-Alcoceba R; Gonzalez-Aseguinolaza G; Mazzolini G; Sangro B; Kramer M G
CS Univ Navarra, Dept Internal Med, Avda Pio 12 36, Pamplona 31008, Spain (Reprint); Univ Navarra, Dept Internal Med, Pamplona 31008, Spain; Univ Navarra, Sch Med, Fdn Appl Med Res, Div Hepatol & Gene Therapy, Pamplona 31008, Spain
jprieto@unav.es
CYA Spain
SO EXPERT OPINION ON BIOLOGICAL THERAPY, (JUL 2004) Vol. 4, No. 7, pp. 1073-1091.
ISSN: 1471-2598.
PB ASHLEY PUBLICATIONS LTD, UNITEC HOUSE, 3RD FL, 2 ALBERT PLACE, FINCHLEY CENTRAL, LONDON N3 1QB, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 243

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ED   Entered STN: 20 Aug 2004
     Last Updated on STN: 20 Aug 2004
     *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*


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     (FILE 'HOME' ENTERED AT 10:41:55 ON 06 NOV 2006)

     INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
     AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
     CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
     DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 10:42:11 ON 06 NOV 2006
               SEA (ADENYLATE(W)CYCLASE) AND (NONSENSE)
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                3   FILE AGRICOLA
                1   FILE BIOENG
                5   FILE BIOSIS
                7   FILE BIOTECHNO
               19   FILE CAPLUS
                1   FILE DRUGU
                1   FILE EMBAL
                8   FILE EMBASE
                1   FILE ESBIOBASE
               19   FILE GENBANK
                4   FILE LIFESCI
                8   FILE MEDLINE
                4   FILE PASCAL
               10   FILE SCISEARCH
                2   FILE TOXCENTER
              197   FILE USPATFULL
               18   FILE USPAT2
L1               QUE (ADENYLATE(W) CYCLASE) AND (NONSENSE)
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     FILE 'SCISEARCH, MEDLINE, CAPLUS' ENTERED AT 10:43:18 ON 06 NOV 2006
L2            37 S (ADENYLATE(W)CYCLASE) AND (NONSENSE)
L3            26 DUP REM L2 (11 DUPLICATES REMOVED)
L4            95 S (ADENYLATE(W)CYCLASE) AND (GENE(W)THERAPY)
L5             0 S L4 AND NONSENSE
L6            13 S (ADENYLATE(W)CYCLASE(W)INHIBI?) AND (GENE(W)THERAPY)
L7          1990 S (GENE(W)THERAPY) AND ARTHRITIS
L8          1426 S L7 NOT PY>2004
L9           979 DUP REM L8 (447 DUPLICATES REMOVED)
L10          638 S L9 AND RHEUMATOID
L11            0 S L10 AND CLITOCINE
L12            4 S (GENE(W)THERAPY) AND (KIDNEY(W)STONES)
L13            2 DUP REM L12 (2 DUPLICATES REMOVED)
L14          206 S (GENE(W)THERAPY) AND (GRAFT-VERSUS-HOST)
L15          134 DUP REM L14 (72 DUPLICATES REMOVED)
L16            0 S L15 AND (ADENYL?(W)CYCLASE)
L17            0 S L15 AND (NONSENSE)
L18            2 S L15 AND (MUTATION)
L19         3159 S (GENE(W)THERAPY) AND (ALZHEIM? OR PARKINSON? OR NEURODEGEN?)
L20            0 S L19 AND (ADENYL?(W)CCLASE)
L21            8 S L19 AND (ADENYL?(W)CYCLASE)
L22            8 DUP REM L21 (0 DUPLICATES REMOVED)
L23          448 S (GENE(W)THERAPY) AND (CIRRHOSIS)
L24          316 S L23 NOT PY>2004
L25            0 S L24 AND (ADENYL?(W)CCLASE)
L26            0 S L24 AND (NONSENSE)
L27          224 DUP REM L24 (92 DUPLICATES REMOVED)

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ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
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